BMS-1

Cat. No.: HY-19991

CAS No.: 1675201-83-8

Molecular Formula: $C_{29}H_{33}NO_5$

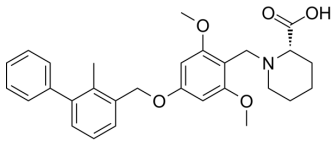
Molecular Weight: 475.58

Target: PD-1/PD-L1; Apoptosis

Pathway: Immunology/Inflammation; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

Methanol : 25 mg/mL (52.57 mM; Need ultrasonic)
DMSO : 7.14 mg/mL (15.01 mM; Need ultrasonic)
DMF : 5 mg/mL (10.51 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.1027 mL	10.5135 mL	21.0270 mL
5 mM	0.4205 mL	2.1027 mL	4.2054 mL
10 mM	0.2103 mL	1.0513 mL	2.1027 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 10 mg/mL (21.03 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1 mg/mL (2.10 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1 mg/mL (2.10 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (2.10 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BMS-1 is an inhibitor of the PD-1/PD-L1 protein/protein interaction (IC_{50} between 6 and 100 nM)[1][2].

IC_{50} & Target

PD1-PDL1[1]

In Vitro

Since PD-1 mediated the exhaustion of natural killer (NK) cell by binding to its ligand PD-L1, BMS-1 (PD-1/PD-L1 inhibitor 1) (1 μM, 3 days) is used to disturb the interaction between PD-1 and PD-L1. Dexamethasone induced increase of PD-1 expression and decrease of cytotoxicity of the co-cultured NK92 cells are reversed by BMS-1[1]. BMS-1, a small-molecule immune checkpoint inhibitor of PD-1/PD-L1, can be used as a therapeutic strategy for tumor immunotherapy[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cytotoxicity Assay[1]

Cell Line:	NK cells and HepG2 cells
Concentration:	1 μM
Incubation Time:	3 days
Result:	Disturbed the interaction between PD-1 and PD-L1.

In Vivo

BMS-1 (500 μg/mL; 100 μL; i.p.) significantly increases the survival rates of the mVEGF165b group and mVEGF165b + MUC1 group[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	BALB/c mice with EMT6 cells[3]
Dosage:	500 μg/mL; 100 μL
Administration:	I.p.
Result:	Increased the survival rates of the mVEGF165b group and mVEGF165b + MUC1 group. mVEGF165b combined with MUC1 results significant retardation of the tumor growth.

CUSTOMER VALIDATION

- Adv Funct Mater. 2021 Mar 21.
- Acta Pharm Sin B. 2023 Aug 19.
- Small. 2022 Nov 11;e2205694.
- J Exp Clin Cancer Res. 2022 Jan 3;41(1):1.
- J Immunother Cancer. 2020 Oct;8(2):e000293.

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REFERENCES

[1]. Zhao Y, et al. Depression Promotes Hepatocellular Carcinoma Progression through a Glucocorticoids Mediated Up-Regulation of PD-1 Expression in Tumor infiltrating NK Cells. Carcinogenesis. 2019 Feb 4.

[2]. Li K, et al. Development of small-molecule immune checkpoint inhibitors of PD-1/PD-L1 as a new therapeutic strategy for tumour immunotherapy. J Drug Target. 2019 Mar;27(3):244-256.

[3]. Zhang H, et al. Utilizing VEGF165b mutant as an effective immunization adjunct to augment antitumor immune response. Vaccine. 2019 Apr 3;37(15):2090-2098.

[4]. Mengyuan Li, et al. KALRN mutations promote antitumor immunity and immunotherapy response in cancer. J Immunother Cancer. 2020 Oct;8(2):e000293.

Caution: Product has not been fully validated for medical applications. For research use only.

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